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<u>L12</u>	l3 with l10	29	<u>L12</u>
<u>L11</u>	l3 near7 l10	4	<u>L11</u>
<u>L10</u>	(carcinoembryonic adj antigen or cea or chorionic adj gonadotrophin)	4654	<u>L10</u>
<u>L9</u>	l3 near4 l4	167	<u>L9</u>
<u>L8</u>	l3 near5 l4	167	<u>L8</u>
<u>L7</u>	l3 near7 l4	171	<u>L7</u>
<u>L6</u>	l3 near8 l4	173	<u>L6</u>
<u>L5</u>	l3 with L4	247	<u>L5</u>
<u>L4</u>	(carcinoembryonic adj antigen or cea or tumor adj antigen or chorionic adj gonadotrophin)	7077	<u>L4</u>
<u>L3</u>	l1 or l2	27259	<u>L3</u>
<u>L2</u>	measles or rindepest or (phocine or canine) adj distemper or (human or bovine) adj respiratory adj syncytial or simian adj virus or sv40 or newcastle adj disease	22516	<u>L2</u>
<u>L1</u>	paramyxoviridae or paramyxovirus or morbillivirus or rubulavirus or pneumovirus or mumps or parainfluenza or sendai	8204	<u>L1</u>

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- ☐ 1. [20020025307](#). 20 Jun 01. 28 Feb 02. Bone sialoprotein based toxic gene therapy for the treatment of calcified tumors and tissues. Koeneman, Kenneth S., et al. 424/93.21; 514/171 514/256 514/263.38 514/44 514/8 A61K048/00 A61K038/17 A61K031/56 A61K031/505 A61K031/522.
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- ☐ 2. [6251365](#). 01 Sep 99; 26 Jun 01. Specific magnetosome, method for the production and use thereof. Bauerlein; Edmund, et al. 424/9.3; 424/130.1 424/450 424/9.1 424/9.2 424/9.321 424/9.34. A61B005/55 A61B009/127.
-
- ☒ 3. [5698530](#). 01 Jul 94; 16 Dec 97. Recombinant virus expressing human carcinoembryonic antigen and methods of use thereof. Schlom; Jeffrey, et al. 514/44; 424/93.2 435/320.1 536/23.1. C12N015/00 C07H021/02 A61K048/00.
-
- ☐ 4. [4729950](#). 29 Jul 85; 08 Mar 88. Enhanced luminescent or luminometric assay. Kricka; Larry J., et al. 435/28; 422/52 435/7.92 435/810 435/968 435/975 436/800. C12Q001/28 G01N053/00 G01N021/52.
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13 near7 110	4

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FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 18:44:28 ON 13 JUN 2003

L1 32265 S PARAMYXOVIRIDAE OR PARAMYXOVIRUS OR MORBILLIVIRUS OR RUBULAVI
L2 67176 S MUMPS OR PARAINFLUENZA OR SENDAI OR MEASLES OR RINDERPEST OR
L3 6174 S CANINE(W)DISTEMPER OR (HUMAN OR BOVINE) (W)RESPIRATORY(W)SYNCY
L4 82085 S SIMIAN OR NEWCASTLE(W)DIESASE OR SV40
L5 165499 S L1 OR L2 OR L3 OR L4
L6 21 S L5(7A) (CARCINOEMBRYONIC(W)ANTIGEN OR CEA OR CHORIONIC(W)GONA
L7 11 DUP REM L6 (10 DUPLICATES REMOVED)

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L7 ANSWER 1 OF 11 MEDLINE DUPLICATE 1
AN 2003228464 IN-PROCESS
DN 22635252 PubMed ID: 12750267
TI Use of a Vaccine Strain of **Measles** Virus Genetically Engineered
to Produce **Carcinoembryonic Antigen** as a Novel
Therapeutic Agent against Glioblastoma Multiforme.
AU Phuong Loi K; Allen Cory; Peng Kah-Whye; Giannini Caterina; Greiner
Suzanne; TenEyck Cynthia J; Mishra Prasanna K; Macura Slobodan I; Russell
Stephen J; Galanis Evanthia C
CS Departments of Neurosurgery [L. K. P.], Molecular Medicine [C. A., K. W.
P., S. G., C. J. T., S. R., E. G.], Pathology [C. G.], Biochemistry and
Molecular Biology [P. M., S. I. M.], and Oncology [E. G.], Mayo Clinic,
Rochester, Minnesota 55905.
SO CANCER RESEARCH, (2003 May 15) 63 (10) 2462-9.
Journal code: 2984705R. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20030517
Last Updated on STN: 20030517
AB Despite the most aggressive medical and surgical treatments, glioblastoma
multiforme remains incurable with a median survival of <1 year. We
investigated the antitumor potential of a novel viral agent, an attenuated
strain of measles virus (MV), derived from the Edmonston vaccine lineage,
genetically engineered to produce carcinoembryonic antigen (CEA). CEA
production as the virus replicates can serve as a marker of viral gene
expression. Infection of a variety of glioblastoma cell lines including
U87, U118, and U251 at MOIs 0.1, 1, and 10 resulted in significant
cytopathic effect consisting of excessive syncycial formation and massive
cell death at 72-96 h from infection. terminal deoxynucleotidyltransferase-
mediated nick end labeling assays demonstrated the mechanism of cell death
to be predominantly apoptotic. The efficacy of this approach in vivo was
examined in BALB/c nude mice by using both s.c. and intracranial
orthotopic U87 tumor models. In the s.c. U87 model, mice with
established xenografts were treated with a total dose of 8×10^7 plaque
forming units of MV-CEA, administered i.v. Mice treated with UV light
inactivated MV, and untreated mice with established U87 tumors were used
as controls. There was statistically significant regression of s.c.
tumors ($P < 0.001$) and prolongation of survival ($P = 0.007$) in MV-CEA
treated animals compared with the two control groups. In the intracranial
orthotopic U87 model, there was significant regression of intracranial U87
tumors treated with intratumoral administration of MV-CEA at a total dose
of 1.8×10^6 plaque forming units as assessed by magnetic resonance
image ($P = 0.002$), and statistically significant prolongation of survival
as compared with mice that received UV-inactivated virus and untreated
mice ($P = 0.02$). Histological examination of brains of MV-CEA-treated

animals revealed complete regression of the tumor with the presence of a residual glial scar and reactive changes, mainly presence of hemosiderin-laden macrophages. In addition, CEA levels in the peripheral blood in both the s.c. and orthotopic models increased before tumor regression, indicating viral gene expression, and returned to normal when the tumors regressed. Ifnar(ko) CD46 Ge transgenic mice, susceptible to MV infection, were used to assess central nervous system toxicity of MV-CEA. Intracranial administration of MV-CEA into the caudate nucleus of Ifnar(ko) CD46 Ge did not result in clinical neurotoxicity. Pathologic examination demonstrated limited microglial infiltration surrounding the injection site. In summary, MV-CEA has potent antitumor activity against gliomas in vitro, as well as in both s.c. and orthotopic U87 animal models. Monitoring CEA levels in the serum can serve as a low-risk method of detecting viral gene expression during treatment, and could allow dose optimization and individualization of treatment.

L7 ANSWER 2 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE
2
AN 2002:471635 BIOSIS
DN PREV200200471635
TI Intraperitoneal therapy of ovarian cancer using an engineered measles virus.
AU Peng, Kah-Whye (1); TenEyck, Cynthia J.; Galanis, Evanthia; Kalli, Kimberly R.; Hartmann, Lynn C.; Russell, Stephen J.
CS (1) Molecular Medicine Program, Mayo Foundation, Guggenheim 18, Rochester, MN, 55905: peng.kah@mayo.edu USA
SO Cancer Research, (August 15, 2002) Vol. 62, No. 16, pp. 4656-4662.
<http://cancerres.aacrjournals.org/>. print.
ISSN: 0008-5472.
DT Article
LA English
AB The use of replicating viruses for cancer therapy (virotherapy) holds much promise. We reported previously that the live attenuated Edmonston B vaccine strain of measles virus (MV-Edm) had antineoplastic efficacy against hematological malignancies. In this study, we demonstrate that a recombinant MV-Edm, genetically engineered to express an inert soluble marker peptide (MV-hCEA), is potent against human epithelial ovarian cancer cells in vitro and in vivo. The virus was selectively oncolytic for ovarian tumor cells but caused minimal cytopathic damage on nontransformed ovarian surface epithelium and mesothelium. In contrast to nontransformed cells, the ovarian tumor cells expressed high levels of the measles virus receptor CD46. When injected directly into large established s.c. SKOV3ip.1 human epithelial ovarian xenografts in athymic mice, the virus induced complete regression of 80% of the tumors i.p. administration of virus enhanced the median survival of mice with advanced i.p. SKOV3ip.1 tumors by >50 days. In addition, we could easily follow the kinetic profile of viral gene expression in the treated mice by determining serum levels of the virally encoded marker peptide (soluble human **carcinoembryonic antigen**). Trackable recombinant **measles** viruses warrant further investigation for therapy of ovarian cancer.

L7 ANSWER 3 OF 11 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 2002:675169 SCISEARCH
GA The Genuine Article (R) Number: 581CK
TI A vaccine strain of **measles** virus genetically engineered to produce **carcinoembryonic antigen** causes regression of glioblastoma multiforme
AU Phuong L K (Reprint); Allen C; Peng K W; Giannini C; Ten Eyck C; Russell S; Galanis E
SO NEUROSURGERY, (AUG 2002) Vol. 51, No. 2, pp. 550-551. MA 725.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0148-396X.

DT Conference; Journal
LA English
REC Reference Count: 0

L7 ANSWER 4 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2002:578948 BIOSIS
DN PREV200200578948
TI Use of a vaccine strain of **measles** virus genetically engineered to produce **CEA** as a novel therapeutic agent against glioblastoma multiforme.
AU Phuong, L. K. (1); Petell, C. (1); Peng, K. W. (1); Russell, S. (1); Galanis, E. (1)
CS (1) Mayo Clinic, Rochester, MN USA
SO Journal of Investigative Medicine, (March, 2002) Vol. 50, No. 2, pp. 176A. <http://www.jinvmed.com/>. print.
Meeting Info.: 2002 Clinical Research Meeting Baltimore, MD, USA April 10-13, 2002 American Federation for Medical Research
. ISSN: 1081-5589.
DT Conference
LA English

L7 ANSWER 5 OF 11 MEDLINE DUPLICATE 3
AN 2001180907 MEDLINE
DN 21105250 PubMed ID: 11160713
TI Single-chain antibody displayed on a recombinant **measles** virus confers entry through the tumor-associated **carcinoembryonic antigen**.
AU Hammond A L; Plemper R K; Zhang J; Schneider U; Russell S J; Cattaneo R
CS Molecular Medicine Program, Mayo Foundation, Rochester, Minnesota 55905, USA.
SO JOURNAL OF VIROLOGY, (2001 Mar) 75 (5) 2087-96.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200103
ED Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010329
AB To redirect the tropism of the vaccine strain of measles virus (MV), Edmonston B, to a targeted cell population, we displayed on the viral hemagglutinin (H) a single-chain antibody (scAb) specific for the tumor-associated carcinoembryonic antigen (CEA). We generated H fusion proteins with three forms of the scAb appended, differing in the lengths of the linkers separating the VH and VL domains and thus in the oligomerization states of the scAbs. All proteins were stable, appeared properly folded, and were transported to the cell surface, but only H displaying the long-linker form of scAb was functional in supporting cell-cell fusion. This protein induced extensive syncytia in cells expressing the normal virus receptor CD46 and also in CD46-negative cells expressing the targeted receptor, human CEA. Replication-competent MV with H replaced by H displaying the long-linker form of scAb was recovered and replicated efficiently in both CD46-positive and CD46-negative, CEA-positive cells. Thus, MV not only tolerates the addition of a scAb on its H protein but also infects cells via a novel interaction between the scAb and its targeted receptor.

L7 ANSWER 6 OF 11 MEDLINE DUPLICATE 4
AN 2000302692 MEDLINE
DN 20302692 PubMed ID: 10842202
TI A transgenic mouse line that develops early-onset invasive gastric carcinoma provides a model for carcinoembryonic antigen-targeted tumor therapy.

AU Thompson J; Epting T; Schwarzkopf G; Singhofen A; Eades-Perner A M; van Der Putten H; Zimmermann W
CS Institute of Molecular Medicine and Cell Research, University of Freiburg, Freiburg, Germany.
SO INTERNATIONAL JOURNAL OF CANCER, (2000 Jun 15) 86 (6) 863-9.
Journal code: 0042124. ISSN: 0020-7136.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200007
ED Entered STN: 20000714
Last Updated on STN: 20000714
Entered Medline: 20000706
AB In an attempt to obtain suitable in vivo models for optimizing new tumor therapy strategies for intestinal adenocarcinomas, **carcinoembryonic antigen (CEA) promoter/SV40 T antigen gene constructs** have been used to generate transgenic mice. One transgenic line (L5496), which contains a 424-bp **CEA promoter/SV40 T antigen transgene**, exclusively developed multi-focal carcinomas in the pyloric region of the stomach in 100% of the offspring. Tumors were already observable in 37-day-old animals as dysplastic cell foci within the mucosal layer. In 50-day-old mice, the tumor mass was mainly restricted to the mucosa with invasive growth into the submucosal tissue. The animals became moribund at 100-130 days of age due to blockage of the pylorus. At this time, the tumor had penetrated into the duodenum and had invaded all tissue layers within the stomach. In contrast to most other stomach tumor models, this one perfectly matches the development of the most common stomach cancers found in humans. Furthermore, after crossing these mice with mice that are transgenic for the human CEA gene, the double transgenic offspring revealed expression of CEA in the resulting tumors. Thus, as well as being a model for studying gastric carcinoma development and prevention, this system should provide a useful preclinical model for CEA-targeted gastric tumor therapy.
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L7 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2003 ACS
AN 1997:472256 CAPLUS
DN 127:159828
TI Expression of transgenic carcinoembryonic antigen (CEA) in tumor-prone mice: an animal model for CEA-directed tumor immunotherapy
AU Thompson, John A.; Eades-Perner, Anne-Marie; Ditter, Margarethe; Muller, William J.; Zimmermann, Wolfgang
CS Institute of Immunobiology, University of Freiburg, Freiburg, D-79104, Germany
SO International Journal of Cancer (1997), 72(1), 197-202
CODEN: IJCNAW; ISSN: 0020-7136
PB Wiley-Liss
DT Journal
LA English
AB Carcinoembryonic antigen (CEA) is a tumor marker for the most common forms of adenocarcinomas. The authors have previously described C57BL/6 mice transgenic for the complete human CEA gene. Compared with humans, these mice reveal a conserved spatiotemporal CEA expression pattern. To establish animal models for CEA-targeted tumor immunotherapy, the authors have crossed CEA transgenic mice with mice that are genetically predisposed to tumor development. These immunocompetent animals should allow optimization of immunotherapy strategies for maximal destruction of tumor tissues with minimal damage to CEA-expressing normal tissues. To develop a breast tumor model, CEA transgenic mice were cross-bred with mice transgenic for the rat neu protooncogene controlled by the mouse mammary tumor virus long terminal repeat. Female offspring developed poorly differentiated breast tumors, none of which, however, expressed

CEA. As a model for colorectal tumors, mice bearing a mutation in the Apc gene (Min mice) and the CEA transgene developed multiple intestinal adenomas with strong CEA expression in all tumor cells. CEA expression had no significant effect on tumor growth. Occasional, well-differentiated breast adenocarcinomas in female offspring expressed CEA focally in tumor cells lining pseudolumina. Crossbreeding ApcMin/+ mice with neu transgenic mice did not reveal a synergistic effect on the kinetics of breast tumor formation. Finally, CEA transgenic mice crossbred with mice transgenic for the SV40 large T antigen regulated by the surfactant protein-C promoter, developed multiple lung adenocarcinomas that revealed a mosaic CEA expression pattern.

L7 ANSWER 8 OF 11 SCISEARCH COPYRIGHT 2003 THOMSON ISI

AN 95:504775 SCISEARCH

GA The Genuine Article (R) Number: RK686

TI TRANSCRIPTIONAL REGULATORY SEQUENCES OF CARCINOEMBRYONIC ANTIGEN - IDENTIFICATION AND USE WITH CYTOSINE DEAMINASE FOR TUMOR-SPECIFIC GENE-THERAPY

AU RICHARDS C A (Reprint); AUSTIN E A; HUBER B E

CS WELLCOME RES LABS, DIV CELL BIOL, 3030 CORNWALLIS RD, RES TRIANGLE PK, NC, 27709 (Reprint)

CYA USA

SO HUMAN GENE THERAPY, (JUL 1995) Vol. 6, No. 7, pp. 881-893.
ISSN: 1043-0342.

DT Article; Journal

FS LIFE

LA ENGLISH

REC Reference Count: 38

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The 5' sequences from the human carcinoembryonic antigen gene (CEA) were analyzed using luciferase reporter gene assays. This analysis identified important cis-acting sequences needed for selective expression in CEA-positive cells. Over 50 CEA/luciferase reporter clones were constructed and analyzed in two CEA-positive and two CEA-negative cell lines. The CEA sequences analyzed extended from the translational start to 14.5 kb 5' of the CEA gene. A 408-bp region from the CEA 3' untranslated region was also examined for its effect on reporter gene activity. The CEA promoter was located between bases -90 and +69 of the transcriptional start site. Sequences between -41 and -18 were essential for expression from the CEA promoter. Multimerization of sequences between -89 and -40 resulted in copy number-related increases in both expression level and selectivity for CEA-positive cells. Two upstream regions of CEA, -13.6 to -10.7 kb or -6.1 to -4.0 kb, when linked to the multimerized promoter led to high-level, selective expression in CEA-positive cell lines. Several CEA/luciferase constructs demonstrated 80- to 120-fold higher expression in CEA-positive cell lines compared to expression in CEA-negative Hep3B cells. The expression from these constructs was quite strong in CEA-positive cells, being two- to four-fold higher than an SV40 enhancer/promoter construct. The most promising CEA transcriptional regulatory sequences were used to regulate the expression of cytosine deaminase (CD) in stable cell lines. The expression of CD was assessed directly by an enzymatic assay and indirectly by determining the in vitro IC50 to 5-fluorocytosine (5FC). The chimeric gene pCEA/CD-145 displayed the desired expression spectrum-high-level expression in the CEA-positive cells and low-level expression in CEA-negative cells. CD expression from this chimera correlated well with the expression of the endogenous CEA gene. Treatment of mice bearing NCI H508 pCEA/CD-145 tumor xenografts with 5FC lead to significant antitumor effects in vivo. The CEA/CD chimeric gene should be useful for tumor-specific suicide gene therapy of CEA-positive tumors.

L7 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2003 ACS

AN 1993:20925 CAPLUS

DN 118:20925

TI Vaccinia virus presenting carcinoembryonic antigen for use in vaccines
 IN Schlom, Jeffrey; Kantor, Judith
 PA United States Dept. of Health and Human Services, USA
 SO PCT Int. Appl., 66 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9219266	A1	19921112	WO 1992-US3843	19920506
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
	CA 2102623	AA	19921107	CA 1992-2102623	19920506
	AU 9220060	A1	19921221	AU 1992-20060	19920506
	AU 674492	B2	19970102		
	EP 584266	A1	19940302	EP 1992-913153	19920506
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, MC, NL, SE				
	JP 06508025	T2	19940914	JP 1992-512025	19920506
	JP 3399943	B2	20030428		
	US 5698530	A	19971216	US 1994-270106	19940701
PRAI	US 1991-695024	A	19910506		
	US 1992-879649	A	19920506		
	WO 1992-US3843	A	19920506		

AB A vaccinia virus presenting carcinoembryonic antigen on its surface by expression of the gene is developed for use in vaccines. The antigen is capable of eliciting an antigenic response in vivo. The gene was introduced into a vaccinia virus vector by std. methods. The gene was expressed in HuTK-143B cells where the antigen was detectable by monoclonal antibodies.. Mice injected with the virus developed a titer to the antigen within 14 days. Mice inoculated with the virus showed a regression of pre-existing implanted tumors; when they were inoculated prior to implantation, there was a dramatic redn. in tumor growth and a delay in onset of growth.

L7 ANSWER 10 OF 11 MEDLINE DUPLICATE 5
 AN 89302087 MEDLINE
 DN 89302087 PubMed ID: 2742579
 TI Expression of an NCA cDNA in NIH/3T3 cells yields a 110K glycoprotein, which is anchored into the membrane via glycosyl-phosphatidylinositol.
 AU Kolbinger F; Schwarz K; Brombacher F; von Kleist S; Grunert F
 CS Institute of Immunobiology, University of Freiburg, FRG.
 SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1989 Jun 30) 161 (3) 1126-34.
 Journal code: 0372516. ISSN: 0006-291X.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 198908
 ED Entered STN: 19900309
 Last Updated on STN: 19900309
 Entered Medline: 19890808
 AB The NCA cDNA, which represents a gene belonging to the CEA family, was inserted into an SV40 early promoter-driven expression vector and used for transfection of mouse NIH/3T3 cells. A cell line, NIH/3T3/KNCA IG7, was selected which expressed a molecule with an apparent molecular weight of 110,000. The mode of membrane attachment of this NCA, which we already proposed to be anchored via glycosyl-phosphatidylinositol, was investigated by treatment of NIH/3T3/KNCA IG7 cells with phosphatidylinositol-specific phospholipase C from Bacillus thuringiensis. Two independent methods, flow cytometry and immunoprecipitation of [3H]-labelled surface glycoproteins, clearly demonstrated that the NCA molecule expressed by NIH/3T3/KNCA IG7 cells is

indeed anchored into the membrane via glycosyl-phosphatidylinositol. Furthermore, these results support our previous biochemical data on NCA-50, by unequivocally showing that the NCA cDNA used for transfection encodes an NCA molecule related to NCA-50 and NCA-90.

L7 ANSWER 11 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1976:202748 BIOSIS
DN BA62:32748
TI CARCINO EMBRYONIC ANTIGEN AND SKIN TEST REACTIVITY IN TUMOR RADIO THERAPY.
AU VIDER M; KASHMIRI R; MOSES B; EARLYWINE D; MEEKER W R; UTLEY J F; MARUYAMA
Y
SO RADIOLOGY, (1976) 119 (3), 677-681.
CODEN: RADLAX. ISSN: 0033-8419.
FS BA; OLD
LA Unavailable
AB Serial carcinoembryonic antigen (CEA) levels were obtained from 122 cancer patients. In a random selection, the levels in 67 of these patients were compared with clinical response to radiotherapy. Skin tests were also performed for histoplasmin, tuberculin and **mumps**. **CEA** levels, skin-delayed hypersensitivity reaction (DHR) and clinical tumor response were evaluated and correlated. Clinical response of tumors to radiotherapy was more often seen in patients with positive skin tests, but no correlation was observed between skin test reactivity and CEA response curves.

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